UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/527,831	01/09/2006	Kathryn Nance North	56182-316187	1014

35657 7590 05/08/2007

FAEGRE & BENSON LLP
PATENT DOCKETING
2200 WELLS FARGO CENTER
90 SOUTH SEVENTH STREET
MINNEAPOLIS, MN 55402-3901

EXAMINER	
POHNERT, STEVEN C	
ART UNIT	**PAPER NUMBER**
1634	

MAIL DATE	DELIVERY MODE
05/08/2007	PAPER

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/527,831	NORTH, KATHRYN NANCE	
	Examiner	Art Unit	
	Steven C. Pohnert	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *12 February 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-16,18 and 24-38* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-16,18 and 24-38* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *15 March 2005* is/are: a)☐ accepted or b)☒ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☒ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *1/19/2007, 3/2/2006, 3/15/2005*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

1. Applicant's election with traverse of group I, 1-24, in the reply filed on 2/12/2007 is acknowledged. The traversal is on the ground(s) that searching all three groups would not cause undue search burden. This is not found persuasive because 371 lack of unity practices are based on unity of invention. As the groups are drawn to mutations in ACTN3, and mutations in ACTN3 are known the inventions do lack a special technical feature over the prior art and thus lack unity of invention. Further the response argues that the invention is drawn to a single gene and thus shares a single inventive concept. This is not found persuasive because each SNP results in a different nucleic acid with different chemical composition sequence and structure. In the instant application the 577R and 577X alleles result in the production of a full-length protein, or a non-detectable protein and as such are distinct. Further applicant traverses the restriction to each SNP and second gene. With respect to the SNP arguments this is not found persuasive for the reasons previously cited. With respect to the selection of a gene this is also found to not be persuasive for in addition to the arguments previously set forth for SNP, each combination of a gene and SNP represents a distinct invention. Each gene encodes a specific protein and thus is distinct from other genes. Further unity had been previously broken with respect to ACTN3 and thus combination of a SNP and gene would also lack unity of invention. However, as newly amended claims 25-28 and 29-32 now depend from claim 1 and are in group I.

The lack of unity is thus maintained, but newly amended claims 25-28 and 29-32 are examined on the merits as they now depend on claim 1.

Newly added claims 33-38 are in group 1 and thus will be examined.

Claims 17,19-23 have been canceled in the 2/12/2007 response.

The requirement is deemed proper and is therefore made FINAL.

A first action on the merits of claims 1-16, 18 and 24-38 follows.

Drawings

2. The disclosure is objected to because it contains an embedded hyperlink and/or other form of browser-executable code. Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01.

Table 3 of the figures contains a hyperlink. The rest of the disclosure must be examined and all other hyperlinks removed.

Specification

3. The disclosure is objected to because of the following informalities:

Page 7, paragraph 0027 of the refers to Table 3 for ethnic differences in the 577 genotype. However Table 3 is a list of SNPs in ACTN3.

Appropriate correction is required.

4. The disclosure is objected to because it contains an embedded hyperlink and/or other form of browser-executable code. Applicant is required to delete the embedded hyperlink and/or other form of browser-executable code. See MPEP § 608.01.

Page 14, line 2 contains a hyperlink. The rest of the disclosure must be examined and all other hyperlinks removed.

Claim Rejections - 35 USC § 112

5. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

6. Claims 1-18 and 24-32 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for determining sprint performance in males by the presence of the ACTN3 577RR genotype, does not reasonably provide enablement for predicting athletic performance in males or females. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

There are many factors to be considered when determining whether there is sufficient evidence to support that a disclosure does not satisfy the enablement requirement and whether any necessary experimentation is undue. These factors have been described by the court in re Wands, 8 USPQ2d 1400 (CA FC 1988). Wands states at page 1404,

"Factors to be considered in determining whether a disclosure would require undue experimentation have been summarized by the board in the Ex parte Forman. They include (1) the quantity of experimentation necessary, (2) the amount of direction or guidance presented, (3) the presence of working examples, (4) the nature of the

invention, (5) the state of the prior art, (6) the relative skill of those in the art, (7) the predictability or unpredictability of the art, and (8) the breadth of the claims."

The nature of the invention and the breadth of the claims:

The claims are broadly drawn to predicting athletic performance in "any" individual based on the presence of one or more variation in ACTN3. "Any " individual broadly encompasses dog, horse, camel, as exemplified in claim 3.

The claims are drawn to the detection of genetic variations "any" ACTN3 gene.

The claims are drawn to the detection of one or more variations in ACTN3 gene.

Claims 5 and 6 further draw the claims to the presence of the 577R or 577RR alleles positively associated with sprinting or power performance.

Claim 8 draws the claims to the 577XX being negatively associated with power performance.

Claim 9 draws the claims to the 577XX being positively associated with endurance performance.

Claim 4 draws the invention to the 1747C>T polymorphism of ACTN3 also known as R577X allele

Claim 18 draws the claims to the detection of at least one other gene.

The amount of direction or guidance and the Presence and absence of working examples.

The specification teaches a study of Caucasian controls and elite athletes comprising 108 endurance and 83 endurance athletes, 88 African Zulu and 152

Australian Caucasian individuals, that elite sprint athletes had a lower frequency of the 577XX genotype of ACTN3 (6% versus 18% in Caucasian population, $p<0.05$)(see paragraph 100 and 102). Thus the specification teaches that elite sprint athletes were less likely to have the 577XX allele than the controls.

The specification further teaches in 46 track athletes competing in events of 800m, 42 swimmers competing in events of 200 m, 9 judo athletes, 7 short-distance track cyclists, and 3 speed skaters. For comparison, a subset of 194 subjects (122 male and 72 female) classified independently as specialist endurance athletes and analyzed, including 77 long-distance cyclists, 77 rowers, 18 swimmers competing over distances of 400 m, 15 track athletes competing in events of 5,000 m, and 7 cross-country skiers. Thirty-two sprint athletes (25 male and 7 female) and 18 endurance athletes (12 male and 6 female) had competed at the Olympic level (paragraph 0103). The specification further teaches "genotypic profiles of the three control groups (150 blood donors, 71 healthy children, and 215 healthy adults) did not differ significantly from one another ($x^2=0.19$; $P=0.996$) nor from a previously genotyped group of 107 white Europeans" (paragraph 104). The specification further teaches there was no significant genotype difference between the elite athletes and control (paragraph 105), although a strong association was seen in sprint athletes relative to controls ($x^2_{[df=5]}=23$; $P<0.001$) (see paragraph 105). The significant allele frequency differences were seen between sprint athletes and controls for both males ($x^2_{[df=1]}=14.8$; $P<0.001$) and females ($x^2_{[df=1]}=7.2$; $P<0.01$) (see paragraph 105). Further the specification teaches that the allele frequencies deviated significantly in opposite directions in the sprint and endurance

athletes (both males ($x^2_{[df=1]}$=13.3; P<0.001) and females ($x^2_{[df=1]}$=5.8; P<0.05) (see paragraph 105).

In example 3 the specification teaches that there is a trend toward significance of the 577XX genotype in endurance athletes, although this reaches statistical significance only in females (see paragraph 110).

In summary the specification teaches that there is no significant difference between elite athletes and controls, although there was a difference in sprint athletes, both male and female. Further the specification teaches there was a significant allele frequency difference between elite endurance athletes and elite sprint athletes.

The specification does not teach any other ACTN3 allele is associated with improved performance. The specification does not teach the R577X allele occurs in any other species.

The state of prior art and the predictability or unpredictability of the art:

The prior art teaches the ACTN3 577X mutation has arisen in humans following the evolution of the ACTN3 gene from primates (Mills et al (Human Molecular Genetics (2001) Volume 10, pages 1335-1346) (see abstract). Mills further teaches that the mouse ACTN3 may not be functionally redundant with the human ACTN3 (see page 1340, 2nd column, 1st full paragraph). Mills further teaches ACTN3 homologs have not been isolated in other mammals (see page 1340, 2nd column, last paragraph). Mills thus teaches that neither the 577X allele nor the ACTN3 gene is predictably found in "any" subject.

Gene card (genecards.org/cgi-bin/carddisp.pl?gene=ACTN3&search=actn3&suff=txt, pages 1-11, 3/24/2007) teaches ACTN3 homologs have only been found in dog, fruitfly, zebrafish, mouse, chimpanzee, and rat (see page 11), while listing 32 species in which a homolog of ACTN3 has not been found. Genecard further teaches there are 9 known SNPs in the human ACTN3 gene and 33 cDNAs. Genecard thus teaches ACTN3 is not predictably present in all species.

Post filing art teaches that the frequencies of the R577X alleles in elite distance runners from Ethiopia and Kenya did not significantly differ from those of their respective control population (see Yang, et al (Med. Sci. Sport and Exercise (2005) volume 37, s42). Yang et al further teaches, "this polymorphism does not contribute significantly to the phenomenal success of elite East African endurance runners."

Moran et al (European Journal of Human Genetics (2007) volume 15, pages 88-93) teaches in a study of 992 adolescent Greeks the presence of the 577R allele resulted in a significant association with sprint times over 40m in males, but not females (see abstract and table 1). However, Moran did not find any other significant correlation of the 577R with tests of power including handgrip strength, basketball throw, vertical jump or agility run (see table 1). Moran further tested aerobic capacity or VO2 max (commonly used tests of endurance performance) and did not see a significant relationship with the 577X allele (see table 1). Moran et al further teaches, "We found no evidence that the R577X genotype is associated with endurance or obesity related genotypes" (see page 93, 1st column last paragraph). Moran thus teaches the 577R

allele is only predictably associated with sprint speed in males. Moran further teaches that the 577R allele is not predictably associated with any other power testing performed. Moran further teaches that the 577X allele is not predictably associated with endurance performance.

Lucia et al studied the frequency of the ACTN3 genotype in a group of 50 top level professional cyclists and 52 Olympic class endurance runners (International Journal of Sports Medicine(2006) volume 27, pages 880-884). Lucia study demonstrated there was no significant differences between the elite runners and cyclists and the R577X genotype. Thus Lucia teaches it would be unpredictable to associate the R577X genotype with improved endurance performance.

The art teaches that presence of SNPs in the same gene does not indicate that each of the genes is associated with the same diseases. Meyer et al. (PG Pub 2003/0092019), for example, teaches that SNPs in the CADPKL gene are not each associated with neuropsychiatric disorders such as schizophrenia. Specifically Meyer teaches that cadpkl5 and cadpkl6 are not associated with the disease, however cadpkl7 has a p-value of less than 0.05, therefore an association exists (see table 5). Each of these polymorphisms are SNPs within the CADPKL gene, however, it is apparent that they are not all associated in the same manner with disease. Thus, Meyer exemplifies that the association of a single SNP in a gene does not indicate that all SNPs within the gene are associated with the disease.

The level of skill in the art:

The level of skill in the art is deemed to be high.

Quantity of experimentation necessary:

In order to practice the invention as claimed, one would first have to establish that a predicative relationship exists between variations in ACTN3 and athletic performance in "any" individual. Experimentation would be replete with unpredictable trial and error analysis because the Mills et al teaches that ACTN3 is not found in all mammals. Mills further teaches this R577X mutation has only been found in humans. Genecard teaches ACTN3 homologs have been identified in dogs, but not camel or horse. Further the specification and claims further limit individual to horse, dog, or camel. Thus the skilled artisan would have to determine if the ACTN3 gene is present in the individual to be screened and further determine if there is one or more genetic variations in "any" individual, then determine if the variations found result in altered performance. This would require undue experimentation to isolate the gene, determine what a variation in the gene is, and correlate the variation to athletic performance.

The skilled artisan would further have to determine if "any" variation in ACTN3 results in altered athletic performance. This would be replete with trial and error experimentation because the art and the specification teach only examines the relationship of the R577X mutation and athletic performance. The art and specification are silent as to the effect of the other 8 known ACTN3 SNPs, as well as the effect of the 33 known 33ACTN3 cDNAs on athletic performance. Further Meyer et al teaches that mutations in the same gene do not result in the same disease or phenotype of improved performance. Thus it would be unpredictable to associate "any" known or unknown

variation in a single gene with a phenotype, even when there is the suggestion that a specific variation may play a role in that phenotype.

The skilled artisan would further have to determine what is encompassed by "any" ACTN3 gene. This would be replete with trial and error experimentation because the ACTN3 gene has not been identified in every species as previously discussed. Further, the ACTN3 has not been identified in 2 of the 4 species claimed. It would thus be unpredictable to associate variations in "any" ACTN3 gene with a phenotype, when the gene has not been identified.

The skilled artisan would further have to determine if 577RR, 577RX, or 577XX are associated with altered or improved sprint or power performance. The specification teaches that all 3 genotypes were found at similar levels in control and elite athletes. As the control population had the same genotypes as the elite athletes the specification teaches genotype is not associated with improved performance. The specification teaches when the elite athletes are divided into sprint athletes and endurance athletes, the male and female sprint athletes significantly different allele frequencies than controls. However, Moran et al found that R577X ACTN3 was positively correlated with sprint performance in males, but not other power specific tests and not at all in females. Thus it would be unpredictable to associate variations in R577X in "any" athletic performance, as the art and the specification have different findings with respect to "power" events and the effect on females and the specification teaches that controls and elite athletes have the same genotype.

The skilled artisan would further have to determine if 577RR, 577RX, or 577XX are associated with altered or improved endurance performance. The specification teaches that the 577XX genotype was slightly higher in endurance athletes. However, Yang, Lucia, and Moran were not able to determine an effect of the 577XX allele in endurance performance of elite runners, elite cyclists, or adolescent children. Therefore it would be unpredictable to use a trend toward significance observed in the specification to associate with improved endurance performance when the post-filing art teaches these findings are unpredictable.

Due to the scope of the claims, one of skill in the art would be required to further undertake extensive trial and error experimentation to determine which individuals possess genetic variation in the ACTN3 gene and determine if any found variations would result in improved athletic performance.

Therefore, in light of the breadth of the claims, the lack of guidance in the specification, the high level of unpredictability in the associated technology, the nature of the invention, the negative teachings in the art, and the quantity of unpredictable experimentation necessary to practice the claimed invention, it would require undue experimentation to practice the invention as claimed.

7. Claims 1-16, 18, 24-38 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter, which was not described in the specification in such a way as to reasonably

convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

The rejected claims 1-16, 18, 24-38 encompass "any" variation in "any" ACTN3. Claim 2 does draw this to human ACTN3, while claim 3 draws to horse, camel, or dog ACTN3. The claims do not set forth any structural requirements for ACTN3.

When the claims are analyzed in light of the specification, the invention encompasses an enormous number of nucleotide molecules. The specification teaches a nucleic acid is from 1 nucleotide up to the whole chromosome. Thus the recitation of ACTN3 encompasses any nucleic acid or any nucleic acid variation on chromosome 11.

In analyzing whether the written description requirement is met for genus claims, it is first determined whether a representative number of species have been disclosed. The instant specification does not teach the sequence of any ACTN3 gene. The specification teaches that, " it is highly probable that a gene like ACTN3 exists in horses but has eluded detection" (see paragraph 0038). The specification thus teaches that applicant did not possess the horse ACTN3 at the time invention was made. Similarly, dog and camel ACTN3 were not taught or described in the specification.

Next, it is determined whether a representative number of species have been sufficiently described by other relevant identifying characteristics (e.g. other nucleotide sequences or positions with in a specific gene or nucleic acid), specific features and functional attributes that would distinguish different members of the claimed genus. In the instant case the specification provides no structural limitation for ACTN3 gene or variations in the ACTN3. The claims read in light of the specification encompass any

nucleic acid molecule that is present on the same chromosome as ACTN3 or has homology with ACTN3 in any species. Further, the claims are drawn to any variation in the ACTN3 gene or the chromosome, which it is on. This is an enormous genus of nucleic acids.

In the instant application, the provided information regarding nucleic acid ACTN3 or any variations, do not constitute an adequate written description of the broad subject matter of the claims, and so one of skill in the art cannot envision the detailed chemical structure of the nucleic acids encompassed by the ACTN3 gene. The claims require genetic variations. The art accepts genetic variations to include transversions, insertions, deletions, translocation, substitutions, and rearrangements. The specification does not describe any insertions, deletions, translocation, substitutions, and rearrangements. Thus the specification does not teach a representative number of species from this enormous genus. Adequate written description requires more than a statement that nucleic acids with a particular quality are part of the invention and reference to a potential method for their identification. The nucleic acid sequence is required.

In conclusion, the limited information provided regarding ACTN3 and its variations is not deemed sufficient to reasonably convey to one skilled in the art nucleic acid molecules encompassed by ACTN3 and its variations.

Thus, having considered the breadth of the claims and the provisions of the specification, it is concluded that the specification does not provide adequate written description for the claims.

Claim Rejections - 35 USC § 103

8. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

9. Claims 1-2, 4-13, 15-16, 24-37 are rejected under 35 U.S.C. 103(a) as being unpatentable over North (Nature Genetics (1999) volume 21 pages 353-354) in view of Costill et al (Journal of Applied Physiology (1976) volume 40, pages 149-153).

North et al teaches the α-actinin-3 (ACTN3) is expressed only in type 2b and 50% type 2a fibers (see page 353, 1st column, last line). North et al further teaches that ACTN3 577XX may be associated with the preferential loss of type 2 fibers (see page 353, 3rd column, last paragraph).

North et al further teaches taking biopsies from an individual (see page 353, column 2).

North teaches genotyping at the ACTN3 locus and for the 1747C>T (R577X) variation and the 1586 A>G (Q523R) (see figure 1e-g)(claims 4, 5, 15, 16, 33, 34, 35, 36). North teaches genotyping by restriction digest results in the determination of the 577RR, 577RX or 577XX genotypes (see figure 1h).

North teaches screening for ACTN3 protein levels with an antibody (see figure 1d) (claims 12, 13, 37).

North teaches fiber typing of a sample (see figure 1d).

North further teaches the force generating capacity of type 2 fibers plays a role in the speed and tempo of movements (see page 354, 2nd column, last3 lines to top of 3rd column.)

North et al does not teach the use of ACTN3 as a marker of type 2 fibers to predict athletic performance. North does not teach selecting an athletic event based on ACTN3 genotypes or the associated fiber type. North does not teach selection of training based on ACTN3 genotypes or associated fiber type composition.

However, Costill et al teaches that training dictates ultimate capacity for endurance, success in sprint or distance running is predetermined by fiber type compositions (see page 153, 1st column, lines 3-6). Costill further teaches that sprint runner had 27.4% slow twitch (type 1) fibers while distance runners had 69.4% slow twitch (type 1). Thus Costill inherently teaches that sprinters thus had 72.6% fast twitch (type 2) fibers while distance runners had 30.6% fast twitch (type 2). Costill thus teaches that increased fast twitch (type 2) fibers is associated with increased sprint performance. Costill teaches determining VO2 Max and fiber type (see page 150, 2nd column 3rd full paragraph).

Therefore it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to combine the genotyping assay of North to screen individuals for the presence of the 577RR or 577RX genotype indicative of type 2 fibers and predict athletic performance with the teachings of Costill that type 2 fibers are related to improved performance in sprinting events. The ordinary artisan would have recognized a prediction could be made between athletic performance and the

577RR genotype. North specifically teaches the 577RR genotype is associated with increased type 2 fibers. Further, Costill teaches fiber type predetermines success in sprint and distance running. The ordinary artisan would further be motivated to base training on the genotype of 577 allele of ACTRN3 because Costill teaches that training dictates ultimate success in endurance events. The ordinary artisan would be motivated to select an event based on the genotype of North as it relates to fiber type because Costill teaches fiber type determines success in sprinting and distance running. The combined teachings of Costill and North would result in better performance by athletes by selecting the event that the athlete is predisposed to do well in and selecting an appropriate training plan.

10. Claim 18 is rejected under 35 U.S.C. 103(a) as being unpatentable over North (Nature Genetics (1999) volume 21 pages 353-354) and Costill et al (Journal of Applied Physiology (1976) volume 40, pages 149-153) as applied to claims 1-2, 4-13, 15-16, 24-37 above, and further in view of Rankinen et al (Medicine in sports and Exercise (2002) pages 1219-2133).

The teachings of North in view of Costill are set forth above.

North in view of Costill does not teach the screening of an individual for a second gene.

However, Rankinen et al teach the screening of 71 loci with respect to performance and fitness phenotype (see abstract). Rankinen specifically teaches that the ACE D genotype is associated with elite Caucasian swimmer in distance of less

than 400m (see page 1220, 2nd column). Rankinen further list in tables 1-7 genes and polymorphisms that are associated with altered exercise performance.

Therefore it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to detect genetic variations in the genes taught by Rankinen in addition to variations in ACTN3 gene taught by North and Costill. One of ordinary skill in the art would be motivated to combine the genes of Rankinen with the ACTN3, because Rankinen teaches the gene recited play a role in exercise performance. Rankinen teaches that the ACE D genotype is associated with elite performance in swimmers. The combination of Rankinen, North and Costill would result in a more accurate prediction of athletic performance.

11. Claim 18 is rejected under 35 U.S.C. 103(a) as being unpatentable over North (Nature Genetics (1999) volume 21 pages 353-354) and Costill et al (Journal of Applied Physiology (1976) volume 40, pages 149-153) as applied to claims 1-2, 4-13, 15-16, 24-37 above, and further in view of Voet et al (Biochemistry, published 199, page 78).

The teachings of North in view of Costill are set forth above.

North in view of Costill does not teach the use of an ELISA.

However Voet et al teaches that Elisa assays using antibodies against the protein of interest are widely used to detect small amounts of protein.

Therefore it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to improve the genotyping assay of North and Costill using an antibody by use of an Elisa. The ordinary artisan would be motivated because Voet teaches this allows detection of small amounts of protein.

12. Claim 14 is rejected under 35 U.S.C. 103(a) as being unpatentable over North (Nature Genetics (1999) volume 21 pages 353-354) and Costill et al (Journal of Applied Physiology (1976) volume 40, pages 149-153) as applied to claims 1-2, 4-13, 15-16, 24-37 above, and further in view of Mills et al (Human Molecular Genetics (2001) Volume 10, pages 1335-1346).

The teachings of North in view of Costill are set forth above.

North in view of Costill does not teach measuring the amount of ACTN3 mRNA in muscle.

However Mills et al teaches Northern blot analysis of ACTN3 to determine expression in mouse tissues (see page 1338, column 1, 1st full paragraph).

Therefore it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to improve the invention of North and Costill to include determining the expression of ACTN3 mRNA in skeletal muscle by northern analysis of Mills. The ordinary artisan would be motivated because Northern analysis allows accurate and quantitative determination of expression of ACTN3.

Summary

No claims are allowed.

Conclusions

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Steven C. Pohnert whose telephone number is 571-272-3803. The examiner can normally be reached on Monday-Friday 7:00-3:30.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on 571-272-0735. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

Steven Pohnert

JEANINE A. GOLDBERG
PRIMARY EXAMINER
3/28/07